Mechanisms of the noxious inflammatory cycle in cystic fibrosis

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Résumé en anglais	Multiple evidences indicate that inflammation is an event occurring prior to infection in patients with cystic fibrosis. The self-perpetuating inflammatory cycle may play a pathogenic part in this disease. The role of the NF-κB pathway in enhanced production of inflammatory mediators is well documented. The pathophysiologic mechanisms through which the intrinsic inflammatory response develops remain unclear. The unfolded mutated protein cystic fibrosis transmembrane conductance regulator (CFTRΔF508), accounting for this pathology, is retained in the endoplasmic reticulum (ER), induces a stress, and modifies calcium homeostasis. Furthermore, CFTR is implicated in the transport of glutathione, the major antioxidant element in cells. CFTR mutations can alter redox homeostasis and induce an oxidative stress. The disturbance of the redox balance may evoke NF-κB activation and, in addition, promote apoptosis. In this review, we examine the hypotheses of the integrated pathogenic processes leading to the intrinsic inflammatory response in cystic fibrosis.
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